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Transthoracic and transvenous pacemakers A comparative clinical experience with 131 implantable units

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This is a review of the clinical experience over a four-year period in treating 119 patients with transvenous and transthoracic pacemakers. Comparison is made of the mortality and complications associated with the two systems, and advantages of both systems are discussed. The one-year survival rate was 78 per cent and 63 per cent for the transthoracic and transvenous systems, respectively. The main complications were related to generator failure, catheter displacement, and electrode fracture. The results indicate that the current pacemaker systems improve the predicted mortality rate for complete heart block, but further improvement in the electronic systems and electrodes is required to correct the frequent complications associated with this mode of therapy.

Since Chardack, Gage, and Greatbatch (1960) and Zoll *et al.* (1961) reported their success with implanted pacemakers, electrical pacing of the heart has become the treatment of choice for symptomatic heart block. The initial pacemakers used epicardial implantation of the electrodes, but Lagergren and Johansson in 1963 reported the use of a transvenous endocardial electrode system. Since that time this type of pacing has become increasingly popular. At the present time, long-term pacing of the heart with implantable pacemaker units may be carried out with either transthoracic myocardial electrodes, or with a transvenous endocardial electrode catheter. The use of myocardial electrodes requires general anaesthesia and a thoracotomy, whereas endocardial electrodes can be implanted using local anaesthesia. The problems associated with each of the techniques may be quite different.

The purpose of this paper is threefold: (1) to describe some of the problems associated with pacemaker therapy, (2) to draw some comparison between the two type techniques of pacemaker implantation currently available, and (3) to provide some preliminary information on demand pacing.

Material and methods

One hundred and thirty-one pacemaker units were implanted in 119 patients who had symptomatic complete heart block with Adams-Stokes seizures or congestive heart failure.

TABLE 1 *Distribution of 131 implanted pacemaker units*

78	Transvenous fixed-rate pacemakers
20	Transvenous demand pacemakers
33	Transthoracic pacemakers
	29 Fixed-rate pacemakers
	3 Synchronous pacemakers
	1 Demand pacemaker
131 units	

Table 1 shows the distribution of various types of pacemaker systems used. Since the inception of the study in 1963, 28 Medtronic[1], one Electrodyne[2], three Synchronous Cordis[3], and one American Optical Demand[4] pacemakers were implanted using the standard transthoracic technique. The ventricular rate before and during operation was controlled with a temporary bipolar catheter in the right ventricle.

Sixty Medtronic and 18 Cordis transvenous fixed-rate pacemaker units have been implanted since 1964. Either the jugular venous system or the cephalic vein was used for the insertion of the permanent transvenous catheter. The electrode

[1] Medtronic Inc., Minneapolis 18, Minnesota.
[2] Electrodyne Co., 30 Endicott St., Norwood, Massachusetts.
[3] Cordis Corporation, Miami, Florida.
[4] American Optical, Bedford, Massachusetts.

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tip was positioned in the right ventricular apex under fluoroscopic control. Satisfactory positioning required that the pacing threshold be less than 1·5 milliamperes. Eleven American Optical and nine Medtronic Demand transvenous pacemakers have been implanted since August 1966, using the same techniques.

The average age of patients with the transthoracic units was 68 years and with transvenous units, 76 years. When this series began in 1963, the transthoracic units were the only type available, and, therefore, patients with these units have been followed the longest.

The transvenous unit became available in 1964, and since then there has been a major shift in our unit preference to this type since it provided a safer means of pacing older and more debilitated patients in whom a thoracotomy would have posed a serious risk. We have continued to use the transthoracic system in the younger patients whom we anticipate pacing for many years, and particularly in children in whom we believe synchronous pacing to be preferable.

Beginning in August 1966, demand pacemakers were implanted. A careful description of the problems encountered with the demand pacemakers will not be considered here, since most of the units were experimental clinical prototype units. There have been a number of competent failures in this group, as would be expected in a new and more sophisticated pacemaker. Though component failures have occurred, there has been no mortality associated with this type of failure.

Results

Duration of pacing and time of death

Table 2 shows the duration of satisfactory pacing and time of death of patients after implantation of the 131 units. There were a greater number of deaths in the transvenous unit in the hospital and during the first six months of pacing. The deaths in the other systems were evenly distributed in the follow-up period.

Transvenous pacemaker experience

(A) *Causes of death* An analysis of the causes of death is presented in Table 3. There were 9 in-hospital deaths occurring in the transvenous group. One of the deaths was related to the pacemaker system in a patient (M.D.), in whom the endocardial pacing threshold increased, and asystole followed by ventricular fibrillation occurred. A second death was probably related to the pacemaker and occurred in patient M.K., who had intermittent heart block with Adams-Stokes seizures. She died suddenly 24 hours after insertion of the transvenous pacemaker. During that 24 hours competition between the patient's normal sinus rhythm and the pacemaker rhythm was observed. Necropsy revealed the catheter to be in good position and no other cause of death was found. The remainder of the in-hospital deaths were related to the severity of the patients' underlying cardiac and metabolic disease.

Of the 13 patients dying after discharge from the hospital, 2 appeared to be directly related to the pacemaker. Patient F.D. had a

TABLE 2 *Survival after pacemaker implantation*

Time after implantation (mth.)	*Transvenous fixed-rate*		*Transvenous demand*		*Transthoracic*	
	Alive	*Dead*	*Alive*	*Dead*	*Alive*	*Dead*
Hospital		8		1		2
0–6	14	6	11	1	3	2
6–12	14	3	5		3	1
12–18	11	3	2		1	1
18–24	7				3	1
24–30	8				3	1
30–36	4				6	1
36–42					1	1
42–48					1	
48–54					2	
Total	58	20	18	2	23	10

FIG. 1 *Necropsy of a patient dying 5 months after permanent transvenous pacemaker implantation, showing endothelialization of the catheter in the right atrium.*

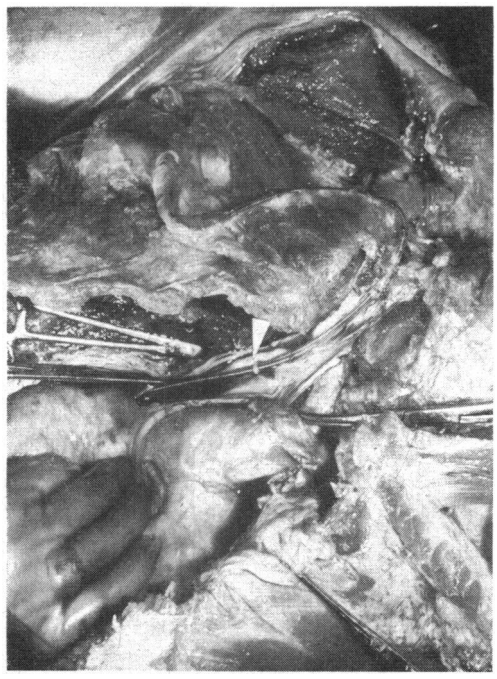


TABLE 3 *Cause of death of 22 patients with transvenous pacemakers*

Patient initials	Age (yr.)	Reason for pacing	Duration	Death				Necropsy findings		
				Due to pacemaker			Clinical cause	Coronary artery disease	Cardiac	Associated findings
				Yes	No	?				
In-hospital deaths:										
R.D.	60	Syncope	24 hr.		×		Hypotension	Moderate	Severe aortic stenosis, LV hypertrophy, fibrosis of interventricular septum	Emphysema and pulmonary fibrosis
M.K.	77	Syncope	24 hr.	×			Unknown	Minimal	Fibrosis of bundle of His; focal myocardial fibrosis	—
T.R.	83	Syncope	4 dy.		×		Azotaemia	Moderate	Fibrosis of posterior interventricular septum; LV hypertrophy	Malignant lymphoma; diabetic glomerulosclerosis
M.B.	66	Syncope	6 dy.	×			Increased pacemaker threshold with failure to pace and ventricular fibrillation	Severe	Apical myocardial infarction; occluded left anterior descending coronary artery	—
G.B.	82	Syncope	7 dy.		×		Cardiomyopathy; low output	No necropsy		
F.M.	79	Congestive failure	11 dy.		×		Septicaemia and liver failure after cholecystectomy	Severe	Thrombotic occlusion of left anterior descending coronary artery; old septal infarction	*Esch. coli* septicaemia, acute hepatitis, and acute colitis
C.F.	85	Congestive failure	16 dy.		×		Pulmonary embolus	Severe	Infarction of interventricular septum and anterior LV; occlusion of left ant. descending coronary artery	Right lower lobe pulmonary infarction and occluded right lower pulmonary artery
A.A.	83	Congestive failure	18 dy.		×		Azotaemia	Mild	Micro-infarct of interventricular septum; cardiac amyloidosis	Systemic amyloidosis
C.M.*	74	Syncope	1 mth.		×		Septicaemia *Esch. coli*	Severe	LV hypertrophy; infarction of anterior and lateral LV wall	Acute pyelonephritis
Out-of-hospital deaths:										
M.D.	78	Syncope	1 mth.		×		Increased angina; congestive failure	No necropsy		
W.B.*	89	Syncope	1½ mth.		×		Cerebral vascular accident	No necropsy		
G.B.	81	Syncope	2 mth.		×		Pneumonia	No necropsy		
L.M.	62	Syncope	2 mth.		×		Congestive failure	Moderate	Old infarction of posterior interventricular septum; ventricular aneurysm, calcific aortic stenosis, and calcified mitral valve	Nephrosclerosis —
N.W.	68	Congestive failure	3 mth.		×		Azotaemia diabetes	Severe	Occlusion of right and circumflex coronary arteries; infarction of posterior LV and interventricular septum	—
J.G.	89	Syncope	5 mth.			×	Unknown	No necropsy		
H.K.	75	Syncope	5 mth.		×		Cerebral vascular accident, unknown	No necropsy		
E.S.	65	Syncope	5 mth.		×		Pneumonia	No necropsy		
A.S.	72	Congestive failure	8 mth.		×		Congestive failure	Mild	Patchy myocardial fibrosis – extensive fibrosis in interventricular septum and conduction system	Infarcts of spleen and kidney

TABLE 3 *Continued*

Patient initials	*Age (yr.)*	*Reason for pacing*	*Duration*	*Death*				*Necropsy findings*		
				Due to pacemaker			*Clinical cause*	*Coronary artery disease*	*Cardiac*	*Associated findings*
				Yes	*No*	*?*				
F.D.	71	Syncope	11 mth.	×			Right atrial thrombo-endocarditis	Moderate	Right atrial mycotic thrombus surrounding pacemaker and obstructing tricuspid valve	Pyelonephritis
G.W.	82	Syncope	12 mth.	×			Competitive rhythm	No necropsy		
J.P.	74	Syncope	13 mth.		×		Multiple pulmonary emboli, acute pyelonephritis	Severe	Subendocardial infarction of LV and papillary muscle	Multiple pulmonary emboli, acute pyelonephritis
R.M.	65	Syncope; congestive failure	15 mth.		×		Congestive failure	Severe	Generalized narrowing of all vessels; calcific aortic stenosis – moderate	—

* Demand transvenous pacemakers.

mass in the right atrium, 2 cm. in diameter, about the catheter partially occluding the tricuspid valve due to monilial thromboendocarditis (Davis, Moss, and Schenk, 1964). Patient G.W., on the day before his sudden death, had severe competition between sinus rhythm and the pacemaker rhythm. The cause of death in patient J.G. was considered questionable, since she died suddenly at home and had been doing well after pacemaker implantation. The remainder of the deaths were considered to be unrelated to the pacemaker; they could be explained either clinically or at necropsy to be due to some other cause.

Of the 22 deaths in the transvenous group, 4 appeared to be related directly to the pacemaker system. The remainder of the deaths were related to severe metabolic or cardiac disease unrelated to pacemaker therapy.

(B) *Non-fatal transvenous complications* The non-fatal complications of the transvenous system are represented in Table 4. The largest number of these complications was related to the instability of the catheter within the ventricle and the difficulty in positioning the catheter without producing an immediate or late perforation. Most of the displacements occurred early, but in two subjects the catheter became displaced after one month. Usually the catheter became fixed to the right atrium and superior vena caval wall within the first 6 weeks (Fig. 1), thus preventing late displacement. We have seen two late fractures in the catheter electrodes. Late perforation occurred in one patient who presented with failure to respond to the pacemaker stimulus (Moss and Rivers, 1966). The perforating catheter could not be removed and a second catheter had to be placed within the right ventricular cavity. In one patient the endocardial system had to be changed to a transthoracic pacemaker unit because of a high pacing threshold of the right ventricular endocardium in excess of generator output. Ventricular fibrillation occurred during implantation in one patient and this was successfully treated and implantation was completed without further difficulty. Phlebitis occurred in the area of the venous catheterization in one patient. Wound dehiscence and erosion of the skin about the generator pocket and the path

TABLE 4 *Non-fatal complications experienced with 78 transvenous fixed-rate pacemaker units*

Catheter complications	
Early displacement (less than 1 month)	13
Late displacement (after 1 month)	2
Wire break	2
Perforation during implantation	1
Perforation—late	1
Increased threshold	6
Generator complications	
Premature generator failure (less than 18 months)	5
Arrhythmia complication	
Ventricular fibrillation during catheterization	1
Miscellaneous complications	
Subclavian thrombophlebitis	1
Wound dehiscence	1
Erosion	2

TABLE 5 *Cause of death of 10 patients with transthoracic pacemakers*

Patient initials	*Age (yr.)*	*Reason for pacing*	*Duration*	*Death*				*Necropsy findings*		
				Due to pacemaker			*Clinical cause*	*Coronary artery disease*	*Cardiac*	*Associated findings*
				Yes	*No*	*?*				
In-hospital deaths:										
M.R.	80	Syncope	12 hr.	×			Post-op. hypotension	Moderate	No myocardial lesions	Diffuse pulmonary fibrosis
L.B.	80	Azotaemia; congestive heart failure	28 dy.	×			Azotaemia; congestive failure	Minimal	Rheumatic mitral and aortic valvulitis (slight) – areas of perivascular fibrosis and hyalinization of septum and ventricular wall	—
Out-of-hospital deaths:										
C.T.	71	Syncope	30 dy.		×		Multiple pulmonary emboli	Minimal	Mural thrombi in right atrium and tricuspid valve – slight narrowing of right coronary artery	Multiple pulmonary emboli
F.V.	76	Syncope; congestive failure	35 dy.		×		Congestive failure	No necropsy		
T.L.	55	Syncope	8 mth.		×		Acute myocardial infarction	Severe	Recent and old thrombus of right and left coronary artery with infarction of anterolateral and posterior LV	Small thromboemboli with organized infarction of left lung
G.H.	77	Syncope	15 mth.			×	Unknown, pacemaker functioning normally	None	Hypertrophy and dilatation of all cardiac chambers	—
C.S.	75	Syncope	19 mth.		×		Cerebral vascular accident	Moderate	Rheumatic heart disease with mitral stenosis and insufficiency, conspicuous narrowing of left and right coronary artery	Subarachnoid haemorrhage
G.K.	67	Syncope; congestive heart failure	27 mth.		×		Congestive failure, azotaemia	Severe	Rheumatic mitral valvular stenosis and insufficiency, thrombotic occlusion of left anterior descending coronary artery, fibrosis and calcification in interventricular septum with absense of conduction system in this area	One small pulmonary arterial thrombosis
E.B.	68F	Syncope	33 mth.		×		Cerebral vascular accident	Moderate	Cardiomegaly without specific myocardial lesions	Cerebral infarction
I.N.	71	Syncope	46 mth.		×		Metastatic carcinoma of colon	No necropsy		

of the subcutaneous catheter occurred in 3 individuals.

Transthoracic pacemaker experience

(A) *Cause of death* There were 10 deaths in the group (Table 5). One patient (M.R.) developed shock and died 12 hours after thoracotomy. A second patient died 30 days after thoracotomy of intractable heart failure. The death of one patient (G.H.) suddenly at home was considered questionably related to the pacemaker, since necropsy failed to reveal any cause of death. He had been in intermittent regular sinus rhythm with pacemaker com-

TABLE 6 *Non-fatal complications experienced with 33 transthoracic pacemaker units*

Electrode complications	
Wire break	3
Increased threshold	5
General complications	
Premature generator failure (less than 18 mth.)	3
Miscellaneous	
Post-pericardiotomy syndrome	2
Battery pocket dehiscence	1
Battery pocket infection	2

TABLE 7 *One-year follow-up of fixed-rate pacemaker units*

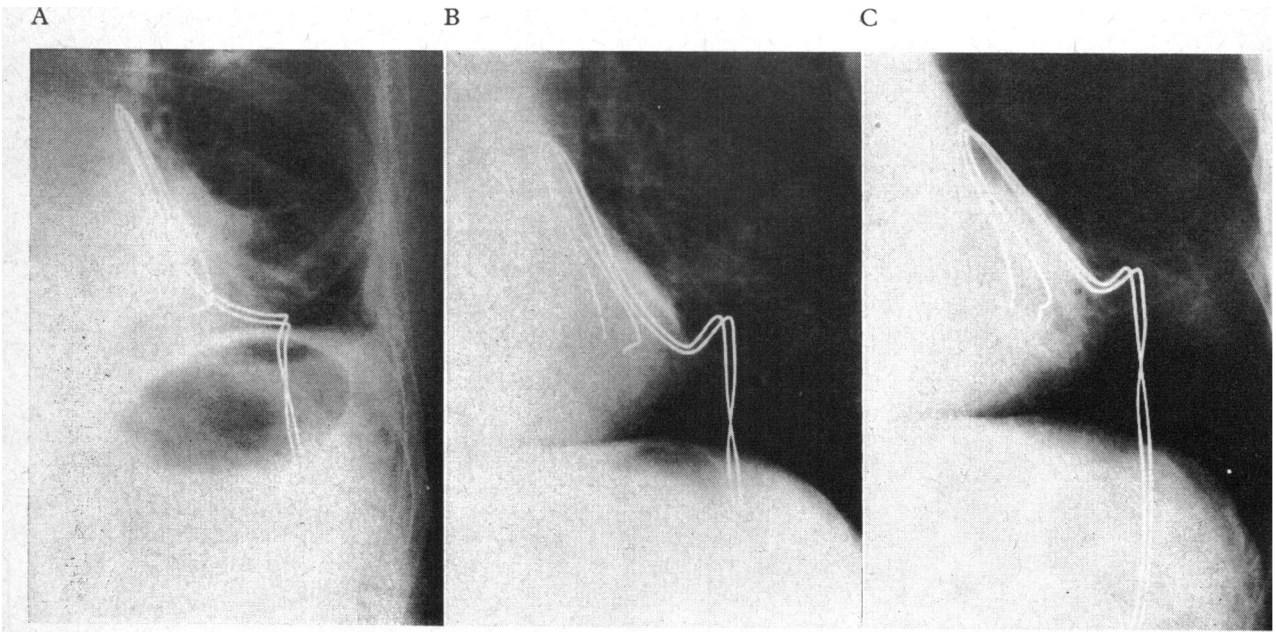
Total		*Living*	*Dead*	*Per cent survival*	*No. of pacemaker related deaths*	*Per cent pacemaker related deaths*
46	Transvenous	29	15	63	4	8·7
23	Transthoracic	18	5	78	2	8·7
69	Total	47	20	70·5	6	8·7

petition. The remainder of the deaths were considered to be unrelated to the pacemaker implantation.

(B) *Non-fatal transthoracic complications* The complications occurring in the transthoracic units are listed in Table 6. In 2 patients, breaks occurred in the extrathoracic course of the electrode. In 1 patient the electrode fracture occurred within the myocardium (Fig. 2). Both patients were eventually changed to the transvenous system. Increased threshold occurred in 5 patients; 4 of these occurred initially when the variable generators were not set at maximum output at the time of implantation. Effective pacing was achieved by increasing the generator output. Subsequently all the adjustable output generators have been set at maximum at the time of implantation. In only one patient did the threshold increase above the maximum generator output, and this required a change to the transvenous system. Generator pocket infection occurred in two patients and was successfully treated by draining the pocket and exteriorizing the generator. Post-pericardiotomy syndrome occurred in 2 patients and was treated with a brief course of steroids.

Comparative one-year survival of both systems A total of 69 units was followed for one year or longer (Table 7). In the 46 transvenous units there were 15 deaths, with

FIG. 2 *Series of x-rays showing fracture of the epicardial leads of a transthoracic pacemaker (A) at the time of implantation, (B) two years later showing a slight bend in one electrode, and (C) four years after implantation showing complete fracture of one electrode and early bending of another.*



a survival of 63 per cent. There were 23 transthoracic units followed for this period, with five deaths and a survival of 78 per cent. We believe that the lower survival rate in the transvenous group was due to the severity of their underlying cardiac and metabolic disease. The definite pacemaker-related mortality in the transvenous group was 8·7 per cent (4/46) and 8·7 per cent (2/23) in the transthoracic group.

Premature generator failure Though the generators had initially been expected to last 4 to 5 years, experience has shown that replacement should be carried out before 24 months. Premature generator failure (less than 18 months of use) occurred in 3 of the transthoracic and 5 of the transvenous units: 6 were Medtronic variable-rate generators and 2 were Cordis fixed-rate generators. Generator failure was usually manifested by change in rate, usually in slowing, but on two occasions by an increase in discharge rate; and was seen as early as 6 months after implantation.

TABLE 8A *Reason for change of system from transvenous to transthoracic*

Patient initials	*Duration*	*Reason*
F.V.	2 hr.	Ventricular perforation with tamponade
A.D.	2 wk.	Fracture of transvenous catheter with stylus still in place
L.D.	4 mth.	Catheter could not be stabilized in huge right ventricle
R.M.	24 mth.	Increased pacemaker threshold

TABLE 8B *Reason for change of system from transthoracic to transvenous*

Patient initials	*Duration (mth.)*	*Reason*
D.D.	7	Increased epicardial threshold
G.S.	12	Wire break
W.M.	23	Wire break
J.W.	28	Myocardial wire fractures

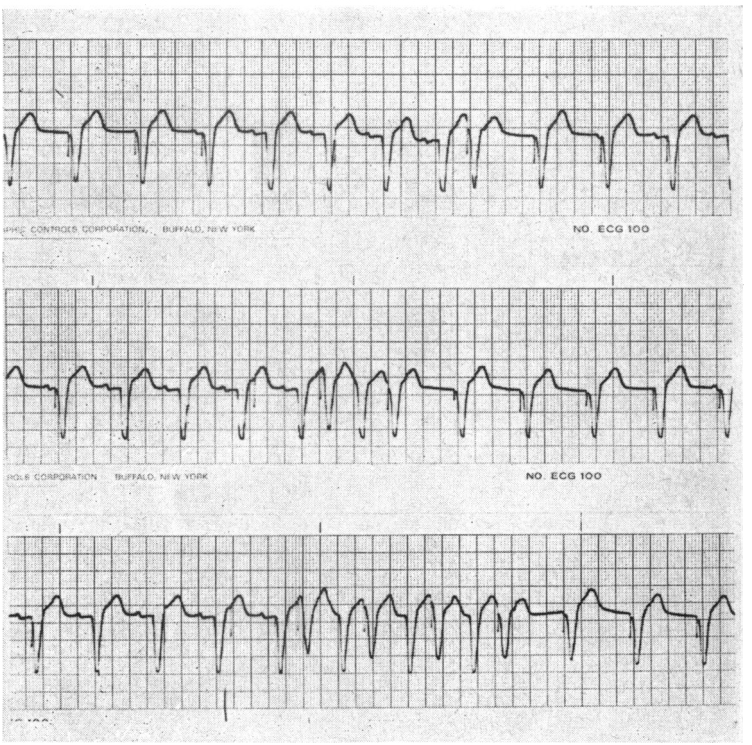
TABLE 8C *Reason for change of system from fixed rate to demand transvenous*

Patient initials	*Duration (mth.)*	*Type*	*Reason*
P.V.	2	Transvenous	Competitive rhythm
L.C.	3	Transvenous	Competitive rhythm
I.S.	22	Transvenous	Competitive rhythm
I.S.	23	Transvenous	Competitive rhythm

Reason for change to different system In 12 patients the type of pacemaker system was changed. The reasons for these changes are listed in Table 8, and some of them have already been discussed. Patient F.V. had a transthoracic pacemaker implanted after he developed acute tamponade due to perforation of the ventricle during transvenous implantation. In patient L.D., with a cardiomyopathy, the right ventricular cavity was very large and initial positioning of the transvenous catheter was quite difficult. When the catheter became displaced at 4 months, a transthoracic unit was implanted. In 4 patients a high degree of competition was noted when the patients returned to regular sinus rhythm (Fig. 3) after implantation, and because of this they were changed to a demand unit without replacing the transvenous catheter.

Incidence of associated cardiac disease Necropsies were performed on 22 of the 32 patients who died. In 9 patients coronary

FIG. 3 *Continuous recording of lead II showing the development of a competitive rhythm. Normal sinus rhythm, occurring with a pacemaker stimulus at a rate of 72, results in a series of interpolated ventricular beats with a resultant rhythm of 150.*



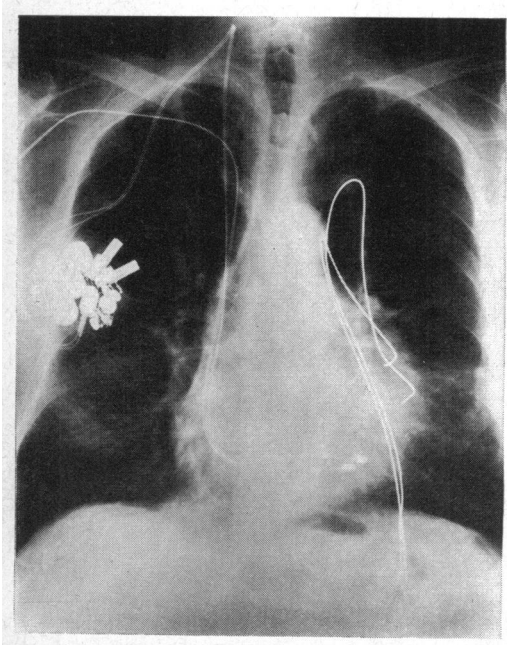
arteriosclerosis was severe and in 8 patients it was moderate and felt to be contributory to the patient's heart block and heart disease. In 5, there was little or no evidence of coronary arteriosclerosis. In 6 patients valvular heart disease was present. Pulmonary emboli occurred in 2 of the transvenous patients and in 3 of the transthoracic patients.

Discussion

Though considerable improvements have been made since the first permanent pacemaker system was implanted by Elmqvist and Senning (1960), many unsolved problems remain. An examination of the data presented in this report leads to certain conclusions regarding the drawbacks and advantages of the current systems and the areas in which further improvements in the pacemaking systems are needed.

There is little question that implanted pacemakers represent the only effective form of therapy for patients suffering from complete heart block and Adams-Stokes attacks. From the time of onset of symptoms the one-year survival reported by Lagergren *et al.* (1966) and Friedberg, Donoso, and Stein (1964) is 50 per cent. In our group of patients, the combined one-year survival is 70·5 per cent. The over-all mortality related specifically to the pacemaker system or to its implantation was 8·7 per cent. Morris *et al.* (1967), in a similar series, experienced a one-year survival of 91 per cent in 44 patients. Chardack *et al.* (1965) reported 71 per cent survival rate after following 50 transthoracic pacemaker patients for two years or longer. Part of our lower survival rate is related to the severe cardiac and metabolic disease associated with heart block in our patients, particularly in the older group treated with transvenous units. The availability of the transvenous system, however, has made pacing available to this group of patients and represents its main advantage. The effectiveness of pacemaker therapy must also be considered in the light of symptomatology and morbidity. With the exception of the period in which non-fatal complications occurred, the patients are almost completely symptom free. Most of the non-fatal complications have been corrected with short periods in hospital and some have not required admittance to hospital.

FIG. 4 *The three separate electrode systems in one patient after two successive failures. Pacing is now accomplished by one of the endocardial electrode systems. The first endocardial catheter could not be removed at the time of its failure when increased threshold occurred.*



The transvenous system has as its major advantage its simplicity in implantation. There are no contraindications to its use. With the exception of the incident of acute ventricular perforation, and one of transient ventricular fibrillation during catheterization, major surgical complications are uncommon. The transvenous system, however, does have certain specific difficulties. Most of these relate to the difficulty inherent in placing the catheter securely within the right ventricle. In some patients there is no good solution, as in patient L.D., who has a large right ventricle secondary to a cardiomyopathy, and in which a catheter could not be stabilized. There is little question that experience in placing the catheter improved our ability to dependably stabilize the catheter, but catheter displacement continues to be a problem which fortunately can be easily corrected.

An increase in endocardial pacing threshold occurred in 6 patients in whom there was no displacement of the catheter. This threshold was greater than the 7 milliamp output of our generators. This occurred as early as 8 months after implantation, and required the placement of a second catheter in a slightly different position. The myocardial threshold usually rises and will stabilize within a few weeks, though significant changes may occur as late as 3 months (Davies and Sowton, 1966; Preston *et al.*, 1966). Preston and Judge (1967) reported a rise in endocardial pacing threshold leading to death in 4½ months. Increased

threshold may be due to infection or improper placement of the electrode, such as in the coronary sinus. In the absence of these causes, fibrosis about the catheter tip is the most likely cause (Siddons and Sowton, 1967). Fractures of the transvenous catheter along with perforation of the ventricle by the catheter also occurred. Six of these catheter problems were complicated by our inability to remove the chronically implanted non-functioning catheter. At necropsy we have seen that fibrous incorporation of the catheter occurs within the atrial wall (Fig. 1) which prevents its removal. In 4 patients we have had to leave all or part of the catheter within the body and particularly within the right ventricle. After a relatively short time, we have 4 patients with 2 pacemaker catheters within the right ventricle (Fig. 4).

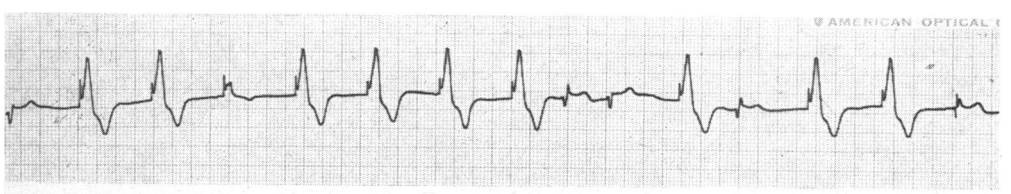
Since pulmonary emboli have resulted from long-term venous catheterizations, we examined our post-mortem data to see if there was an increased incidence of pulmonary emboli in the transvenous group. Though pulmonary emboli were found, there was no apparent increase of emboli in the transvenous as compared to the transthoracic group.

The main difficulty with the use of the transthoracic system lies in the risk of thoracotomy to these patients who for the most part were quite elderly. Our operative mortality would be even higher if we did not have the option to use the transvenous system. Both of the operative deaths occurred in patients before the availability of transvenous systems, and in patients who would have been candidates for the transvenous units under our current criteria for system selection. Electrode fractures did occur and repair did not provide long-term continuity. The fracture of the intramyocardial portion of the electrode could be corrected by converting the system to a unipolar system. If both electrodes fractured, it was necessary to change to a transvenous system. If a bend occurred during implantation of the myocardial electrode, fracture ultimately resulted. In 2 patients, in spite of seemingly perfect initial placement, a bend occurred and fracture later developed, probably due to intramyocardial stresses (Fig. 2).

Generator dysfunction occurred in both transvenous and transthoracic with equal frequency. Generator failure can be due to dysfunction of any one of its components or to battery wear-out. Failure was usually manifested by change in pacing rate, though total cessation of pacemaker discharge did occur. This may be due to decreased energy output of the generator, but is usually due to component failure. One of the main causes of this type of failure is penetration of body fluid as a vapour into the epoxy casement leading to a component failure. Other failures were related to fatigue of the components themselves. It is clear that the original prediction that the generators would last five years was unfounded. We have elected to change units after two years, but because failure can occur at anytime, we obtain monthly electrocardiograms on all our patients. As the electrical components improve, generator wear-out will become more of a problem, and longer lasting or rechargeable batteries will be required.

Our experience with the demand units is relatively brief, as compared to the longer and larger follow-up of the fixed rate systems: we believe it adds further flexibility in pacemaker selections and added safety to pacemaker therapy. Sowton and Davies (1964) noted that in patients with normal sinus rhythm and pacemaker parasystole, the death rate was five times greater than in those patients with total pacemaker capture. Bilitch, Cosby, and Cafferky (1967) also reported a higher mortality using permanent endocardial pacing in patients with competitive rhythm. Studies in both animal and man have shown the hazard of electrical stimulation of the heart in the vulnerable period of the T wave. Usually the

FIG. 5 *Demand pacemakers function in a patient with sinus bradycardia with syncope. Demand pacemaker discharge occurs only after a delay of 72 msec. after a sinus discharge, and continues to discharge until a sinus beat occurs. The ninth beat is a sinus beat followed by an atrial premature contraction. The fourth and fifteenth beats are fusion of the sinus and paced beats occurring simultaneously.*



energy required to produce ventricular fibrillation is much greater than the stimulus of the pacemaker generator (Chardack, Gage, and Greatbatch, 1961). Other investigators have shown in animals that various factors such as quinidine (Rothfeld *et al.*, 1965), hypoxia, and coronary ligation (Nathan, Medow, and Pina, 1965) will lower the fibrillation threshold. Castellanos, Lemberg, and Berkovits (1966), using a transvenous catheter, were able to generate multiple extrasystoles and ventricular fibrillation in humans when the impulse fell close to the peak of the T wave, using energy of less than four times the stimulus threshold for contraction. We believe that two of the deaths in our series may have been related to competitive pacing, and we have changed to a demand generator when competition occurs.

Demand pacemakers have been recently developed (Castellanos *et al.*, 1966; Zuckerman *et al.*, 1967) which sense the R wave and produce a pacemaker discharge only when a prescribed RR interval is exceeded, thus functioning like an escape pacemaker and preventing stimulation of the ventricle in the vulnerable period (Fig. 5). We are now using this type of demand generator as the system of choice in patients who have Adams-Stokes attacks with intermittent periods of regular sinus rhythm.

The permanent transvenous pacemaker represents an important addition to the pacemaker therapy. Demand pacemakers also have provided an additional new safeguard to permanent pacing. In spite of these advances, examination of our experience indicates that certain improvements in our current systems are needed. Improvement in the generator is required in view of the relative frequency of component failure. More reliable and longer-lived generators should be within the reach of our electronic capability. The occurrence of a failure to pace due to increased myocardial threshold can be corrected by providing a generator with a higher energy output, particularly for the transvenous system where this appears to be a more significant problem. The development of a more durable endocardial and epicardial electrode will further decrease the number of complications.

The two types of implanted systems complement each other and enable us to offer pacemaker therapy to a wider variety of patients. Pacemaker therapy in individual patients must be viewed over a long period. We must look forward to providing pacemaker therapy to patients for 20 and 30 years and in children even longer. Using our present equipment, this will require 10 to 15 generators and an unknown number of electrode changes during this period of time. Because of this, we have chosen to start with a transthoracic system in all young patients. When the patient is older, we may then change to a transvenous system, if necessary. In spite of these difficulties, it is clear, however, that the currently available pacemakers are the most dependable means of treating chronic heart block.

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References

Bilitch, M., Cosby, R. S., and Cafferky, E. A. (1967). Ventricular fibrillation and competitive pacing. *New England Journal of Medicine*, **276**, 598.

Castellanos, A., Lemberg, L., and Berkovits, B. V. (1966). Repetitive firing during synchronized ventricular stimulation. *American Journal of Cardiology*, **17**, 119.

Chardack, W. M., Gage, A. A., Federico, A. J., Schimert, G., and Greatbatch, W. (1965). Five years' clinical experience with an implantable pacemaker: an appraisal. *Surgery*, **58**, 915.

—, —, and Greatbatch, W. (1960). A transistorized, self-contained, implantable pacemaker for the long-term correction of complete heart block. *Surgery*, **48**, 643.

—, —, and — (1961). Correction of complete heart block by a self-contained and subcutaneously implanted pacemaker. *Journal of Thoracic and Cardiovascular Surgery*, **42**, 814.

Davies, J. G., and Sowton, E. (1966). Electrical threshold of the human heart. *British Heart Journal*, **28**, 231.

Davis, J. M., Moss, A. J., and Schenk, E. A. (1969). Tricuspid candida endocarditis complicating a permanently implanted transvenous pacemaker. *American Heart Journal*, **77**, 818.

Elmqvist, R., and Senning, A. (1960). An implantable pacemaker for the heart. In *Medical Electronics (Proc. 2nd Int. Conference on Medical Electronics, Paris, June, 1959*, p. 253. Ed. by C. N. Smyth. Iliffe, London.

Friedberg, C. K., Donoso, E., and Stein, W. G. (1964). Nonsurgical acquired heart block. *Annals of the New York Academy of Sciences*, **111**, 835.

Lagergren, H., and Johansson, L. (1963). Intracardiac stimulation for complete heart block. *Acta Chirurgica Scandinavica*, **125**, 562.

—, —, Schüller, H., Kugelberg, J., Bojs, G., Alestig, K., Linder, E., Borst, H. G., Schaudig, A., Giebel, O., Harms, H., Rodewald, G., and Scheppokat, K. D. (1966). 305 cases of permanent intravenous pacemaker treatment for Adams-Stokes syndrome. *Surgery*, **59**, 494.

Morris, J. J., Jr., Whalen, R. E., McIntosh, H. D., Thompson, H. K., Brown, I. W., Jr., and Young, W. G., Jr. (1967). Permanent ventricular pacemakers. Comparison of transthoracic and transvenous implantation. *Circulation*, **36**, 587.

Moss, A. J., and Rivers, R. (1966). Myocardial perforation by a permanent transvenous pacemaker. *New England Journal of Medicine*, **275**, 265.

Nathan, D. A., Medow, A., and Pina, P. (1965). Ectopic cardiac response to electrical stimulation. *Circulation*, **32**, Suppl., p. 159.

Preston, T. A., and Judge, R. D. (1967). High myocardial threshold to an artificial pacemaker—Report of a fatal case. *New England Journal of Medicine*, **276**, 797.

—, —, Bowers, D. L., and Morris, J. D. (1966). Measurement of pacemaker performance. *American Heart Journal*, **71**, 92.

Rothfeld, E. L., Zucker, I. R., Parsonnet, V., and Lotti, L. (1965). Effect of quinidine on competitive cardiac pacing. *Circulation*, **32**, Suppl. 2, p. 182.

Siddons, H., and Sowton, E. (1967). *Cardiac Pacemakers*, p. 170. Charles C. Thomas, Springfield, Illinois.

Sowton, E., and Davies, J. G. (1964). Investigations of failure of artificial pacing. *British Medical Journal*, **1**, 1470.

Zoll, P. M., Frank, H. A., Zarsky, L. R. N., Linenthal, A. J., and Belgard, A. H. (1961). Long-term electric stimulation of the heart for Stokes-Adams diseases. *Annals of Surgery*, **154**, 330.

Zuckerman, W., Zaroff, L. I., Berkovits, B. V., Matloff, J. M., and Harken, D. E. (1967). Clinical experience with a new implantable demand pacemaker. *American Journal of Cardiology*, **20**, 232.